Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Physical Activity and Coronary Artery Disease

TO THE EDITOR: The Medical Staff Conference "Exercise and Coronary Artery Disease—What Should We Be Recommending to Our Patients (and Ourselves)?"[1] is a timely and well-organized assessment of the topic. It reflects awareness of the controversies, importance relative to coronary mortality and remarkable contemporary public participation.

Whereas few physicians consider habitual physical activity status as an important component of the medical history, we believe it is. A very simple but effective distinction is whether, how often and how regularly persons engage in sufficient dynamic endurance exercise to develop an endogenous heat load to initiate sweating for several minutes in ordinary ambient temperature and humidity environments. The Seattle Heart Watch studies classified subjects as "active" if they engaged in such endurance exercise at least once per week, regularly each week. The subsequent incidence of coronary disease events in this group was lowered to the same extent as observed in other epidemiologic studies utilizing various techniques to study the effect of habitual physical activity (Bruce, unpublished observations, 1985).

A pilot study suggested that symptom-limited exercise testing motivates persons to alter remediable risk factors by changing health habits.[2] It apparently was more effective in cardiac patients with demonstrable impairment who subsequently intervened against excessive body weight and smoking.

The physiologic adaptations induced by exercise conditioning are primarily an increase in functional aerobic capacity (maximal oxygen intake or $\dot{V}O_2$ max) and a slight, but often sufficient, reduction in *relative* aerobic requirements, heart rate and blood pressure at any given level of submaximal or ordinary physical activity, and consequently increased cardiac and aerobic reserves. Normally this is achieved by an increase in stroke volume and widening of the arterial-mixed venous oxygen difference. In coronary patients only the peripheral component of this adaptation is observed, and this results from an increase in arterial oxygen content during exertion to further widen the arterioventricular oxygen difference.[3] The locus of the peripheral effect is in the conditioned skeletal muscles.

The adverse effects of exercise in high-risk persons include sudden cardiac arrest or death. Risk factors for this event have been identified.[4] The Jim Fixx syndrome of "clinically silent coronary artery disease" with sudden death is more appropriately designated the *denial* of exertional discomfort which he had experienced for weeks before the event.

In the Seattle population more than 40% of asymptomatic healthy men have none of the conventional risk factors. Exercise testing has not been of predictive value in these men; nevertheless, such testing defines circulatory performance and functional capacity.[5] In contrast, exercise testing of healthy men with any one or more conventional risk factors shows about 1% prevalence of any two or more of four abnormal responses to exercise[6]; this prevalence increases with age up to 3% in men of 60 or more years. In these high-risk men the subsequent incidence of primary coronary heart disease events is significantly greater and comparable to the incidence of "hard" secondary events (that is, myocardial infarction or cardiac death) in men known to have coronary heart disease.

Of particular interest in relation to the aging processes, rates of decline in Vo_2 max with increasing age vary with source of data—that is, cross-sectional versus longitudinal measurements.[7]

From a societal viewpoint of an aging population, high cost of chronic illness, especially in older patients, and increasing need for medical cost containment, the possible role of an active life-style in *postponing* the onset of symptomatic chronic cardiovascular disease represents a hypothesis that warrants intensive studies for the next generation.[8] Since some evidence indicates little change in life expectancy, this may result in "compression of morbidity," which may contribute to cost-containment of chronic illness.[9]

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Get Boxing Off TV

TO THE EDITOR: The AMA and the California and New York state medical associations have called for a ban on boxing, but attempts to obtain enabling legislation have failed. I suggest a new approach.

The Surgeon General has declared that smoking is harmful to health. As a result, cigarette commercials are prohibited from television. TV revenues are the lifeblood of professional boxing. I propose that an effort be made to have the Surgeon General declare that boxing poses an unusual threat to the health of the participants. Consequently, TV broadcasts of boxing events are directly contributing to a clear health hazard and should be prohibited.

A case could also be made that such displays of "legitimate" extreme violence may contribute to violent behavior of others, particularly of young persons.

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Diabetes and Pregnancy

TO THE EDITOR: The relationship between diabetes and pregnancy management illustrates the continuing need for practitioners to update their knowledge. There are more than 11 million known diabetic persons in the United States and many more undiagnosed. Some 3% of all pregnant women are affected by diabetes.

Medical management of possibly pregnant diabetic patients consists of screening, preconception counseling and monitoring, outreach and education, active patient participation, regular prenatal visits and utilization of improved maternal and fetal surveillance.

In 1909 maternal mortality was 30% and fetal loss 65% in diabetic patients. Since the discovery of parenteral insulin in 1921, pregnancies have increased. In the past few years maternal mortality has been all but eliminated and maternal morbidity reduced significantly. Perinatal mortality from stillbirth, prematurity and birth injury for insulin-dependent gravidas approaches that of normal gravid women.

During the past five years significant changes in management have occurred. Historical and clinical "clues" (family history, obesity, glycosuria, large baby and the like) are not enough to diagnose the possible gestational diabetic as we move towards screening every prenatal. At the very least, all pregnant women 25 and older should be screened. Home monitoring includes reflectance meters for accuracy and a discussion on changes of diet to high fiber, more carbohydrates and calculation of calories on "ideal" body weight. Glycosylated hemoglobin and ultrasound are no longer experimental but are proposed for regular four and six weeks' prenatal monitoring purposes. Use of daily unconjugated urinary estriols is no longer optimal management. β-Mimetic agents used to control premature labor, like commonly used ritodrine, are contraindicated with diabetes, and magnesium sulfate becomes the preferred method of tocolysis. PG (phosphatidylglycerol) replaces L/S (lecithin-sphingomyelin ratio) for the assessment of fetal lung maturity. And postpartum, even fewer obstetricians now recommend even low-dose oral triphasic contraception to young insulin-dependent diabetic women. Where the risk for a diabetic woman bearing an infant with a birth defect is 3 to 13 times greater than for a nondiabetic woman, the emphasis is on good health, preconception control and euglycemia in pregnancy for a very much improved perinatal outcome.

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Neuropsychological Assessment

TO THE EDITOR: In the October 1985 issue, Drs Morton and Linz properly called attention to the role of neuropsychological testing in assessing the degree and pattern of cognitive impairment associated with toxic encephalopathy from chronic exposure to toxic solvents.[1]

Dr Markovitz suggested, in a letter to the editor in the January issue,[2] that many of these cases arise after a company closes and are then a subject of litigation. He disputed Drs Morton and Linz. He suggests such testings "are highly variable, especially in Workers' Compensation cases." He pointed out varying conclusions offered by different psychologists administering testing. He indicated that there may be many nonspecific findings. He called attention to a woman who was tested while distracted by a young baby in her lap. He suggested that neuropsychological testing alone can lead to "overdiagnosis."

Since I refer patients for neuropsychological assessment in cases of possible encephalopathy associated with chronic exposure to organic solvents or trauma, I believe this is an important issue. In my opinion, Dr Markovitz introduces "red herrings." He emphasizes that different psychologists may offer different conclusions. Certainly this is no different than different cardiologists interpreting electrocardiograms in varying ways, or radiologists differing as to the interpretation of particular radiographs. He complains of nonspecific findings. This is little different than a large plurality of electrocardiogram reports.

Dr Markovitz anecdotally cites a psychologist who concluded that a patient had brain damage when she was tested while holding a baby in her lap. Certainly I have seen cases where diagnostic studies were misinterpreted due, for example, to inadequate electrode jelly on the electrocardiographic electrode, improper placement of a patient before the x-ray machine or other misapplications of technique.

Dr Markovitz seems to throw out the baby with the bath water. Neuropsychological testing is indeed a valuable diagnostic procedure, just as are electrocardiograms, radiographs and other processes. Neuropsychological testing should not be condemned because of the fatuousness Dr Markovitz describes, just as all electrocardiograms and x-ray studies